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ALCOHOLISM—A NATIONAL PUBLIC HEALTH EMERGENCY*

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The title of this paper is an exaggeration only if one confines public health narrowly to questions of communicable disease and sanitation—in other words to a somewhat out-of-date concept. The developing pattern of life on this planet is going to necessitate a new orientation in the sphere of activity of a public health department. There will in fact be no sense in continuing to disregard the large amount of public ill-health that exists, but which is not caused by communicable disease nor has anything specifically to do with food, housing or sanitation. This particular kind of public debility is going to be the problem of the future, and health administrators will be forced to take cognisance of it. Already it is with us on a large scale, and the increasing addiction of the people to alcohol, especially in the lower strata of society, is merely symptomatic of a growing inability on the part of these people to live with any care or thought for the welfare of their fellows and especially for their own family circle. This indifference to the welfare and respect of the family unit is of sinister augury for the future. The State cannot afford a big rupture on this front. Already social services are straining every nerve to bolster the position up.

If, however, nothing can be done about the demoralised human soil in which alcoholism so readily grows, public health authorities will have to put some energy into dealing with the alcoholism itself. Up to the present, however, they have taken rather a detached view of this, as if it were a matter outside their province. This state of affairs must not be allowed to continue because until Public Health departments can see how essentially alcoholism falls within their sphere as a cause of much human illness and mortality, the sooner they will begin to use their influence with the Government to take the necessary steps to deal with it. At the moment it is nobody's baby. To the Department of Justice it is merely a matter of punishment if the law is transgressed. There are a number of charitable organisations which do what they can, but this does not touch the main body of the trouble.

We have now reached a stage when the bill in terms of money and misery which this country has to meet for its alcoholism is no longer a laughing matter. No longer is there need for the items of this account to be set out in all their distressing details of homes wrecked and children mentally damaged. We need not ask the Department of Justice what they spend annually on a police force that appears never to be adequate to cope with the Union's crime sheet, and we know that a great part of that crime sheet comes directly or indirectly from alcohol. Venereal disease also thrives in an alcoholic environment.

Why therefore make so much fuss over a few cases of communicable disease when the really big public health issue today concerns the demoralisation, with its associated alcoholism, which is overtaking large sections of South African society. The State cannot go on paying for health and social services, not to mention police control, when these services are being continually undermined by a pattern of life into which alcoholism easily fits and each year tends to produce a darker social picture, and more alcoholism.

That brings me down to the main purpose of this paper—namely, the Bantu people of Natal. Here we have an extraordinary state of affairs, in so far as these people are becoming heavily addicted to alcohol, and

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yet one hears practically nothing about it from official sources. The Bantu race have now got such a taste for strong drink, which is none other than a craving for intoxication—that I doubt whether any force could now make them give it up. This has nothing to do with the drinking of comparatively harmless Kaffir beer. One of the most important reasons, and one not often mentioned, why natives no longer want to work on farms, is the call of strong alcohol that can now be acquired in urban areas. The higher wages obtainable there are admittedly part of the reason, but the attraction of strong drink is a large part of the incentive, and yet this fact is being repeatedly missed or overlooked. When once a native has experienced the intoxication of either shimiyane or gabeen, he may return to the farm for a while, but it will not be be for long.

When therefore, one looks at a shack settlement on the fringe of a large urban area, by all means think of the high wages of industry, and the changing outlook of the native, as cause for its existence, but do not overlook the part played by the growing addiction of these people to strong alcohol. When a public health official says that these settlements spread tuberculosis, dysentery and venereal disease, he does so chiefly because of the overcrowding and the lack of proper sanitation. In most cases, however, you will find no mention in his report of alcohol. By overlooking the part played by alcohol in the spread of V.D. and in lowering the resistance to diseases like tuberculosis he is missing half the evil of these settlements. Why does he not mention alcohol? He knows how much liquor the police capture when they make a raid. He must know how alcohol acts on the nutrition by its process of substitution. He must know that money spent on alcohol means less money spent on proper food, and less money sent home for the support of the family back in the kraal.

If there was an outbreak of poisoning by a chemical such as arsenic, public health authorities would become extremely energetic to bring it to an end. But alcohol is also a chemical, the toll from which in terms of human illness, misery and death in one year in this country puts any other outbreak of poisoning into insignificance, but for some reason or other it has never been considered a public health matter. Obviously the developing pattern of life on this planet will require a changed public health outlook in the near future.

Reverting to the shack settlement, one often hears it stated that if the shacks could be replaced by better accommodation and better sanitation, the greater part of the evil of these settlements would disappear overnight. It is wishful thinking to assume that better housing is going to have the effect of reducing the consumption of this type of alcohol. It will not do so, because the two are unrelated in spite of much opinion to the contrary. The true underlying reason is that the Bantu people are now going down into a state of demoralisation, through frustration and the loss of the steadying effect of tribal law. The rising against the Indians was partly an attempt by them to recover some racial face or conceit, but they had to be shot down for it by the white man, so back to the alcohol they have gone with a natural feeling of despair. Take heed, a fine race, a race that is almost the life blood of the country, is being subjected to a circle of forces that is rotting them physically and morally. They see no hope for themselves, and growing numbers are finding solace in strong drink.

Unfortunately a complication has lately arisen which makes the whole matter more serious. It is that the native has learnt how to make a liquor even stronger than shimiyane. He has learned the art of distillation, and in the native reserves has the necessary privacy and opportunity to distil shimiyane into the much more potent gabeen. Now here is a first-class public health issue. Gabeen drinking is spreading rapidly through the reserves, and even the women are becoming addicted to it. The result of this is two-fold, firstly, resistance to tuberculosis and acute amoebic dysentery is being grievously lowered, and secondly, the children are being neglected. We have already been told authoritatively that the death-rate and incidence of tuberculosis among these people are increasing. Moreover, the incidence of the fulminant type of acute amoebic dysentery among Bantu adults, especially the men, is intimately connected with the consumption of strong drink. The number of these cases which are treated at King Edward VIII Hospital in Durban annually gives an idea of the magnitude of the problem, but the part in causation played by gabeen and shimiyane is never stressed or even mentioned. About one out of every three natives in Natal is a carrier of amoeba histolytica. This condition they carry with very little symptomatology, until they become addicted to shimiyane or gabeen. The whole infection is then lit up, and an urgent hospital case is sooner or later produced. Go into one of these hospitals and notice the type of native who has this kind of illness. It is with few exceptions not a woman, nor is it a child or young adult. But it is the native man, the type who gets some solace from intoxication. Heavy tough labour on a poor nutritional substrate, is also a factor, but strong alcohol is the precipitator.

Now what are the health centres doing about this heavy gabeen drinking in the reserves? The answer is, nothing, because the position is almost untouchable except by the police, and they are at two grave disadvantages. Firstly the size of the Force is too small for work of this kind, and secondly native look-outs in the reserves are too efficient for police surprise. All the police can do is to make a periodic raid on a shack settlement in an urban area when they usually recover enough liquor to float the proverbial battleship. Heavy fines are paid, but in two or three days, the position has reverted to where it was before.

The neglect of children in the native reserves was mentioned above as one of the results of the introduction of gabeen. The very young native child has never had a great deal of care taken of him by his father from time immemorial. The native mother has had to do this as best she could, besides her duties of tending the mealies or other crops, and fetching the water. When her husband goes off to find work in a city, she looks forward to getting a pound or two from him to help feed the family. City life, however, soon changes him in several fundamental respects, the net result of which is to reduce each month the amount of money he sends home. He begins

to spend it on drink and European clothes. Intoxication in an urban area often leads him to contract venereal disease. His wife back at the kraal keeps carrying on as best she can, and the school feeding of native children in the reserves has been a Godsend to her. But the husband has made it an excuse for sending her less money. Unfortunately the picture today is of heavy gabeen drinking in the reserves, with increasing addiction on the part of the women as well.

Imagine therefore the plight of the young children, and consider how important it is that at this particular juncture the school feeding of the children should not only be maintained but increased. This is a public health matter, though some, with an eye on the economic future of the country might think that it came under a different heading. In any case public health and economic interest are intimately bound up. The point, however, that one wishes to make is that the nutrition of these people, never very good, not only of the children, but also of the parents is being further reduced as a consequence of the advent of gabeen drinking in the reserves. Couple this with the heavy consumption of shimiyane in urban areas, and you begin to get an explanation of why tuberculosis incidence and the death-rate are mounting in these people, and why it is that they seem to be peculiarly addicted to acute amoebic dysentery. The latter is not a racial predisposition. It is merely a question of the power of this type of alcohol to undermine the nutrition and to lower the whole resistance of the bowel mucous membrane to the inroads of the parasite.

Reverting now to the general question of alcoholism among the different races in this country, it would appear that the bulk of the trouble is coming from that section of the population which has a somewhat low intelligence. Admittedly there is much too much alcoholism among certain groups of educated Europeans who ought to know better, the type for instance who make a habit of frequenting night-clubs, but the upper and professional classes, as a whole, can usually keep alcohol in its proper place, and would not be very averse to total prohibition were such considered necessary in the national interest. This class is the backbone of the country and to use a colloquial expression, 'can take it' in many respects, when asked to do so. One is not advocating total prohibition, but to a large extent the main burden of expense in connection with alcoholism in this country is borne by this same group, and if it is their advocacy that the sale of alcohol to, or its production by, those of low intelligence, be drastically restricted, a wise government should pay heed to it. When I use 'its production by' I am referring more particularly to the Bantu natives in Natal.

Every man has his coefficient of inertia, so do countries and communities. We are up against State inertia, because naturally the required drastic action on this front will affect the livelihood of a large number of people, and the number of people who make a living out of alcohol, both dietetically and financially is extremely large. The State hesitates therefore to step in. A country can be moved to action by the threat of war, or the threat, on a large scale, of civil insubordination, but it is only with the greatest difficulty that one can interfere with the community's way of life for what are termed 'lesser considerations.' It would, however, be interesting to know how much further the ravages of alcohol have to go in this country before responsible people are brought to realise that there is need for action on a war-emergency level. It seems therefore that the time has come when we as a responsible profession must either sit back and watch with fiendish and detached interest the whole social structure of this country being degraded by this pernicious chemical, or we must think up a stimulus that will act like a war emergency, to bring those responsible to say 'Hold! enough, this is where we take drastic action'.

As a matter of practical politics what can be done to diminish the toll of alcoholism on the national purse and its ravages on public health? The restriction on length of these papers make it impossible to deal with so large a question here. One or two moves however, do suggest themselves. In the first place it should be enquired whether the health centres are not merely browsing on the outskirts of larger and deeper trends, the debilitating causes of which they can never hope to catch up with. If that is due to the focus of their activity being somewhat ill-directed, then the sooner it be altered the better both for reasons of urgency on the economic front, and because the incidence of certain diseases is showing no response to their present activity. In the second place, it is beyond dispute that enormous quantities of sugar are being sold into the reserves by the local stores. How much of this sugar is being used for gabeen production, should be the subject of an enquiry. A possible answer to this question may be obtained by a reasonable *per capita* requirement for nutrition and the density of population. An obvious excess of sugar-purchase might very well be found, in which case it might be advisable to put some of these stores on a quota. It is felt, however, that we do not know nearly enough about what goes on in the fastnesses of these reserves. European policemen will ride in when they want to arrest someone, or collect evidence of crime, and the district surgeon will go in if the local magistrate orders him to do so, but whether the crime is due to alcohol, or whether resistance to disease is being undermined by alcohol, seems to be nobody's concern.

A large number of cases of injury and illness among natives, are continually pouring into Provincial hospitals from the reserves, and from urban areas. Most of the cases of injury have arisen in fights where the combatants are intoxicated. In regard to the cases of illness, it would be worth an enquiry to find out, even if only approximately, what proportion of them had been aggravated or precipitated by indulgence in liquor of the shimiyane or gabeen type.

Our native problem today may therefore not be merely a growing exodus from the farms to industrial centres, and from a public health point of view, not merely a question of communicable disease, but the larger question of whether the native is not getting more and more out of hand in a disciplinary sense, and whether his growing addiction to alcohol is not only the cause of this, but is also the underlying cause of much of the burden which Provincial hospitals are carrying for the treatment of native illness today.

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VAN DIE REDAKSIE

SCHISTOSOMIASE: 'N ONLANGSE OORSIG

'n Ondersoek van bilharsiose as 'n geheel, wat grotendeels op patologiese en kliniese ondervinding gegrond is, is nog nooit in Afrika buitekant Egipte aangepak nie. Die treffendste werk in Egipte in hierdie verband is dié van Fairley oor proefondervindelike besmetting van ape, wat hy in 1920 gepubliseer het.

Elders in Afrika is kleiner verhandelinge oor besondere of beperkte kliniese onderafdelings van schistosomiase deur verskeie skrywers, veral in Suid-Afrika, bygedra. Geen van hierdie skrywers het egter 'n beeld van die kliniese en patologiese verskynsels van die siekte as 'n geheel gegee nie.

Dr. M. Gelfand* het (in 'n onlangse monografie) gepoog om die siekte, soos dit die verskillende organe aantas, so omvattend as moontlik te skets en sy verhandeling maak waarskynlik die eerste poging uit om in die algemeen die klinies-patologiese aspekte van schistosomiase wat in Suidelike Sentraal-Afrika gesien word, te beskryf. Die skrywer se ongeëwenaarde geleenthede om alle aspekte van hierdie siekte eerstehands te bestudeer, het hom in staat gestel om 'n buitengewone en omvattende beeld daarvan te gee.

Bilharsiose het vandag ernstige afmetings in baie dele van die Unie van Suid-Afrika en byna die hele Oos- en Sentraal-Afrika aangeneem. Bloot om hierdie rede is hierdie verhandeling oor bilharsia-siekte vir alle geneeshere belangrik, met inbegrip van diegene wat met die verskillende voorbehoeddienste op die vasteland van Afrika besig is.

Nie net is die literatuur oor die kliniese en patologiese aspekte van schistosomiase uiters breedvoerig bespreek nie, maar sekere waarnemings word te boek gestel in die lig van die uitgebreide ondervinding wat deur die skrywer in verband met hierdie siekte in Suid-Rhodesië verkry is. Aan ons word dus die geleentheid gegee om bilharsiose te ken soos die patoloog of die klinikus sifilis, melaatsheid of tuberkulose ken. Die monografie bring die patologiese en kliniese eienskappe van 'n toestand wat in sy openbarings uiters veranderlik is, baie duidelik in verband.

Belangrike gevolgtrekkings is die bewys dat bilharsiose nie tot vatbaarheid vir kanker aanleiding

* *Schistosomiasis in South Central Africa: A Clinico-Pathological Study.* By Dr. Michael Gelfand, M.D., M.R.C.P. (Pp. 239. 25s.) Published for the Post-Graduate Press by Juta and Company, Limited, Cape Town and Johannesburg.

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EDITORIAL

SCHISTOSOMIASIS: A RECENT REVIEW

A study of bilharziasis as a whole, based largely on pathological and clinical experience, has not been attempted in Africa outside Egypt. The most impressive work in Egypt in this connexion is that of Fairley on the experimental infection of monkeys, which he published in 1920.

Elsewhere in Africa, smaller publications dealing with particular or restricted clinical branches of schistosomiasis have been contributed by various authors, notably from South Africa. None of these writers, however, has given a picture of the clinical and pathological appearances of the disease as a whole.

Dr. M. Gelfand* has (in a recent monograph) attempted to provide as broad an outline as possible of the disease as it affects the various organs and his publication probably represents the first attempt to describe in general the clinico-pathological aspects of schistosomiasis seen in South Central Africa. The author's unrivalled opportunities to study all aspects of this disease at first hand have made it possible for him to produce an outstanding and comprehensive picture of the disease.

Bilharziasis has to-day reached serious proportions in many parts of the Union of South Africa and almost the whole of East and Central Africa. For this reason alone this account of bilharzial disease is important for all practitioners including those engaged in the various preventive services on the African continent.

Not only is the literature on the clinical and pathological aspects of schistosomiasis discussed very fully, but certain new observations are recorded in the light of the extensive experience gained by the author about this disease in Southern Rhodesia. We are thus afforded an opportunity to know bilharziasis as the pathologist or the clinician knows syphilis, leprosy, or tuberculosis. The monograph correlates very clearly the pathological and clinical features of a condition very variable in its manifestations.

Important conclusions are the demonstration that bilharziasis does not predispose to cancer, especially in

gee nie, veral nie in die geval van die urine- en spysverteringskanale en die lewer nie. Getuienis word ook aangevoer om te bewys dat bilharsiose nie 'n oorsaak van sirrose is nie.

Die werk handel grotendeels oor die kliniese eienskappe van bilharsia-siekte en die skrywer se omvangryke ondervinding van na-doodse ondersoeke het hom in staat gestel om die simptoomleer met die patologiese uitwerkings in verband te bring.

Die publikasie van dr. Michael Gelfand se monografie oor schistosomiase is 'n belangrike mylpaal in die geskiedenis van geneeskundige skryfwerk op hierdie vasteland. Dr. Gelfand het talryke bydraes tot die kliniese literatuur oor tropiese siektes gelewer en is die skrywer van 'n baie geslaagde ondersoek, *The Sick African* wat al reeds twee uitgawes beleef het. Die huidige werk behels die navorsing wat dr. Gelfand vir sy M.D.-graad aan die Universiteit Kaapstad onderneem het.

Die uitgewers moet geluk gewens word met die beskikbaarstelling van hierdie uitstekende bydrae tot die studie van tropiese siekte.

the case of the urinary and intestinal tracts and the liver.

Evidence is also adduced to show that bilharziasis is not a cause of cirrhosis.

The work deals mainly with the clinical features of bilharzial disease, and the author's extensive post-mortem experience has allowed him to correlate the symtomatology with the pathological effects.

The publication of Dr. Michael Gelfand's monograph on schistosomiasis marks an important event in the history of medical writing on this continent. Dr. Gelfand has made numerous contributions to the clinical literature on tropical diseases and is the author of a very successful study entitled *The Sick African* which has already gone into two editions. The present work represents the research which Dr. Gelfand undertook for his M.D. degree at the University of Cape Town.

The publishers are to be congratulated on making available this distinguished contribution to the study of tropical disease.

DIAGNOSIS AND TREATMENT OF POLIOMYELITIS* IN THE ACUTE STAGE

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The diagnosis of poliomyelitis in a patient immediately imposes upon us many and varied responsibilities, both in regard to the community at large and the patient in particular. The earlier such diagnosis is established the sooner may we meet these responsibilities. The following remarks are based upon personal experience in the 1945 and 1948 epidemics on the Witwatersrand when an opportunity was afforded for examining an unfortunately large number of cases.

How are we to recognise the illness in its early stages? At times this is easy since there may be sufficient circumstantial evidence to warrant suspicion of the disease, though no individual sign or symptom could in itself confirm the diagnosis. At times, however, there may be few signs to label the illness as such. We must remember that the majority of cases may be said to be almost subclinical in their manifestations and only during epidemics are we likely to suspect a number of these. The occurrence in a family of one case of clinically-recognisable poliomyelitis would compel us to view with great circumspection any minor illness in that household at more or less the same time; we should insist on complete bed rest in such an instance, thus possibly preventing the illness, if it is poliomyelitis, being aggravated into one of severity.

Some years ago we were apt to regard the disease as appearing dramatically—'the child awakened paralysed' are the actual words of some of our earlier lecturers. Insufficient emphasis was laid on the prodromal period despite the observations of Wickman and others who stressed the extreme rarity with which paralysis appeared without any preceding symptoms. Whereas formerly diagnosis awaited the appearance of paralysis, we can now quite frequently recognise pre-paralytic or non-paralytic poliomyelitis with reasonable assurance and we find that paralysis is, in fact, a sequel in only a small number of instances.

Many of these mild cases are referred to as being abortive, but the term is often used too loosely to indicate all those cases of infection with the virus in which there is no consequent damage to the cord or brain. This designation should be reserved only for those patients with no meningeal involvement, who suddenly in the course of sound health develop headache, fever, vomiting, possibly tachycardia and rapidly become normal within 24 to 48 hours. They reveal no rigidity of spine or neck, have a normal C.S.F. and in fact present symptoms scarcely sufficiently characteristic to justify a definite

* This paper was read at the Medical Congress of the Association held at Cape Town in September 1949.
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diagnosis. They should rather be called suspected abortives, and can only be regarded as possible cases of infection during epidemics or where frank cases are met with in the household at the same time.

Of the clinically-recognisable cases most may be labelled as a meningeal form of the disease. The greaer number of these do not develop paralysis and thus may be said to have had the non-paralytic type. In those eventually developing paralysis the same phase would be labelled the pre-paralytic stage. Whether paralysis develops or not, this phase is coloured by signs and symptoms of meningeal involvement but other symptoms suggestive of a systemic infection may occur as well. Fever, headache, malaise and a generalised muscular aching may all initiate the condition. To these are sometimes added either signs of a gastro-intestinal disturbance such as nausea, vomiting or even diarrhoea or an upper respiratory infection such as coryza, sore throat or actual tonsillitis.

What picture do we see when we first examine the child, usually in the household? Our patient appears anxious or fearful, suspicious of approach, unduly alert. He may be somewhat tense and pre-occupied and resents interference. His face is flushed, often with a circum-oral pallor resembling that of scarlet fever and he tends to perspire freely, especially on the brow. There may be drowsiness from which state he is easily aroused only to manifest apprehension.

The mother may relate that the child has had disturbed sleep for several nights, attended with bad dreams or even nightmares. He has been restless, possibly irritable, and the mother may say that she has never seen her child in such a peculiar state with any previous illness. He may have complained, possibly only once, of a pain in his back or one or other limb. Not uncommonly the urine has been held for an unusually long period and a bed-wetter may have had his first dry night. This retention may persist for days and thus provide considerable discomfort. Whereas diarrhoea may have been present initially the child usually soon becomes constipated.

The pulse is rapid, often disproportionate to the degree of temperature which is frequently not above 102°-103°. Voluntary movements may seem unsteady and the whole body may be tremulous. If we allow him to walk across the room we note his peculiar hesitant gait, a little unbalanced, and if we ask him to turn suddenly he almost falls. He is possibly giddy and certainly anxious to get back to bed. He is unwilling to sit and seems to sidle up to this posture fearing it will hurt. He supports himself with his arms outstretched to the rear (tripod sign). He is glad to lie down again and may like to curl up on his side.

Moderate neck rigidity may be present but especially remarkable is the so-called 'spinal rigidity', which is in fact a resistance to anterior flexion of the neck and spine and can be easily demonstrated by the chin-knee test, or in milder cases by an inability on the part of our patient to approximate his chin to the chest without evoking great discomfort. He may reveal a certain clumsiness or awkwardness in turning over from the back on to the abdomen while lying flat in bed, owing to the spinal stiffness. The Kernig sign may be positive but we must beware of misinterpreting normal inability to flex the leg to a right angle at the hip. In young babies spinal rigidity cannot be detected so easily by the usual measures but the mother may have noted that on lifting the infant by the legs in order to change the napkin, the child will cry, whereas normally it does not resent this manoeuvre. At times the meningeal signs are pronounced and may last for days or even weeks.

There is hyperaesthesia to light touch and muscle tenderness may be evident. If he is an older child he may complain of pins and needles or may state that his limb does not seem to belong to him. The reflexes at this stage are usually increased. Vasomotor disturbances of the skin such as flushing, mottling, tache or even rashes may be observed.

He may reveal peculiar sweeping movements of the eyes or he may demonstrate nystagmus. This is frequently present, either in lateral or vertical fixation; it is usually horizontal but sometimes of the rotatory type.

If we do a blood examination this will probably reveal only a mild leucocytosis and a normal or only a slightly raised erythrocyte sedimentation rate.

The non-specific components of the illness are sometimes referred to as the first stage, the signs and symptoms of invasion of the nervous system as the second stage, since the latter do not usually appear until after the second day or so. It will be noted that these two stages may be present together or may follow closely on each other, almost insensibly shading from one stage into the other.

Occasionally, however, they may be separated by a very clear-cut interval, the so-called 'diphasic' or erroneously labelled 'dromedary' type. 'Camel' type would be a more appropriate name as there are two 'humps' of temperature. In this variety the initial temperature may settle after three to four days in what has resembled an abortive attack or some other illness. The child has possibly already been allowed up, somewhat injudiciously, one might add, during a poliomyelitis epidemic; then, several days later there is a secondary rise in temperature. There follow all the accompanying signs of meningeal irritation and the full tragedy is now possibly upon us.

Here we have depicted the clinical set-up so frequently seen during the epidemic last year. The diagnosis could only be confirmed by examination of the C.S.F. or by the onset of paralysis if this was destined to occur.

The cerebro-spinal fluid is usually under only slight pressure, 120-140 mgm. water, is clear to the naked eye though it may present a faint haze under transmitted light and occasionally is frankly clouded. The cell count reveals a greatly varying pleocytosis of 20 to 800+, usually about 50 to 250 cells, polymorphonuclears predominating in the early stages but being rapidly displaced by lymphocytes. Within several days, sometimes earlier, the cell count drops considerably and in those patients developing paralysis the cell count may be insignificant at the time palsy ensues. The total protein is increased during the meningeal involvement and in fact usually rises while the cell count drops, persisting often for several weeks. The sugar and chlorides are usually within normal limits.

This whole clinical picture gradually subsides in the

non-paralytic cases and in doing so never follows exactly the same pattern. Some patients may feel completely well within a few days, exhibiting no clinical signs, whereas others may not be relieved of a number of their symptoms for many days or even weeks. The so-called 'meningitic symptoms', often suggesting nerve root irritation are particularly liable to persist for many days. On the other hand the initial symptoms may be part of a pre-paralytic phase and then usually on about the fourth to the fifth day, sometimes later or even many days later, occasionally earlier, paralysis may supervene.

It is customary to divide the paralytic types of the disease into spinal, spinal-respiratory and bulbar forms, but it must be appreciated that combinations of these usually occur. Paralysis as such generally begins suddenly and achieves its full extent rapidly, but occasionally does so with a more protracted course, progressing in small spurts over a period of several days. Typically it is flaccid, associated with loss or depression of the reflexes and with hypotonicity. It is said that the legs are more frequently involved than the arms, but this was not appreciably the case in our 1945 and 1948 epidemics on the Witwatersrand. There seems to be a definite predilection for certain muscule groups, such as the deltoids and anterior tibials. Muscles supplied by the same spinal segments may demonstrate considerable variation in the degree of this paralysis. Unless paralysis is absolutely complete in both corresponding limbs it is most unusual to have a truly symmetrical picture. Paresis may be so minimal, at times, as to be easily overlooked.

Muscle spasm frequently develops but it took an epidemic to impress upon most of us with what extent and severity this may occur in one or more groups of muscles. It is not truly a spasm as one would understand an upper motor neuron lesion but rather a 'muscle tightness'. Muscles in spasms are painful to stretching or pressure and may often appear weak or even completely immobile; if we force passive movement we induce pain. The chief factor in causing disability is paralysis, but spasm contributes to both deformity and disability. When the opposing muscle is in painful spasm and shortened, deformity will result. In addition, if parts are left for some time in a deformed position, secondary myostatic contraction develops, fixing the deformity. This in turn inhibits muscle function and with it, the return of muscle power. Muscle tightness may be extensive and disabling and yet there be little basic muscle weakness. At other times there may be a good deal of paralysis in muscles other than those in spasm; disappearance of the spasm then reveals the extent of the flaccid paralysis.

Reference has already been made to the urinary retention in the pre-paralytic phase and this may persist for several days after the onset of a paralysis.

Intestinal paralysis may also occur with a consequent tendency to abdominal distension and constipation, still further aggravated by a not uncommon abdominal muscular paralysis. In the latter instance the palsy is best recognised by a localised bulging of the abdominal wall when the child cries, coughs or attempts to sit up. Paralysis of the rectal sphincter is extremely rare.

Respiratory failure may occur through involvement of the muscles of respiration, particularly the intercostals and the diaphragm. Because of their closely-approximated nuclei in the spinal cord, the nerves to the shoulder girdle and the phrenics are often conjointly affected. There is thus frequent association of diaphragmatic palsy with deltoid weakness, especially if both deltoids are severaly paralysed. Spasm of these muscles may also occur and interfere with their function.

Bulbar involvement is not usually difficult to recognise. There is generally considerable fever, marked prostration and a much more toxic appearance than in the spinal forms. In this brain-stem type of the disease the motor cells of the cranial nerves are affected. In this variety bulbar symptoms sometimes develop without the general pre-paralytic signs. For example, in several cases difficulty in swallowing or a nasal twang of the speech were the first symptoms noted by the parents. All combinations of cranial nerve paralyses may be seen, varying from affection of a single cranial nerve to extraordinary multiple palsies. Respiratory failure may be brought about by involvement of the respiratory centre in the medulla or by affection of the appropriate cranial nerves (including the motor fibres of the glossopharyngeal).

Myocarditis is now known to occur in poliomyelitis. The E.C.G.'s done on a number of our cases would make interesting discussion but there is no time at our disposal to consider this subject.

Space permits only mere mention of the encephalitic, ataxic and Landry forms of the disease, but careful watch should be kept for the possible development of these.

What common mistakes may be made in diagnosis? During an epidemic the mass hysteria is so great that a considerable proportion of the patients suspected by their relatives or themselves of having contracted the disease have scarcely anything wrong with them, let alone poliomyelitis. Doctors are bound to make many errors in suspecting this illness when none is eventually found to exist, making due allowance for abortive cases. Passing reference should at least be made to a number of the pitfalls presenting in the path of diagnosis and which we personally encountered.

Muscular Weakness. During an epidemic it is easy to be misled into regarding weakness associated with fever consequent upon any infection or contagious disease, as being the result of poliomyelitis. Due care must be exercised not to lose our true sense of proportion and the history and clinical findings must be analysed with discretion.

Myopathies and myotonias must similarly be excluded.

Rheumatic Fever. This is quite often confused with poliomyelitis. In epidemics it is more usual to find rheumatics labelled poliomyelitis, since everyone's attention is focussed on this condition. In sporadic cases it would be more likely to find a poliomyelitis case wrongly diagnosed as rheumatic. Examination must attempt to establish whether the patient is really unable to move or whether he resents movement because of pain. If the latter, is the pain due to hyperaesthesia or muscle spasm or is it caused by joint pathology? Apart from other clinical findings a sedimentation rate would be helpful

in the differential diagnosis as this is only slightly, if at all, raised in poliomyelitis whereas in acute rheumatic states it is usually high.

Other arthritic conditions may lead to similar confusion, particularly that variety common to young children manifesting itself usually in one joint only and consequent upon some local infection, generally of the throat. The pain will cause the limb to be kept immobile but careful examination exposes the nature of the condition.

Adenitis. Cervical adenitis, especially if deep to the sternomastoid muscles, may deter the child from moving his head freely, owing to the pain, and this may easily be mistaken for nuchal rigidity. Lateral and rotatory movements are usually more painful than forward flexion of the head when these enlarged glands are present. Deep inguinal adenitis may be painful and result in the patient refusing to move the leg. During the epidemics referred to many children affected with adenitis in these sites were wrongly diagnosed as poliomyelitis.

Meningitis. In the early stages it may be difficult to differentiate meningitis, whether pyogenic or tuberculous, from a meningitic phase of poliomyelitis. As a rule pyogenic meningitis is accompanied by a more pronounced pyrexia and rigors not infrequently occur. The child is usually more acutely ill than in poliomyelitis but not necessarily so. In tuberculous meningitis it is generally found that indefinite symptoms have been present for at least two to three weeks, whereas in poliomyelitis the prodromal period is commonly of short duration. In meningitis the neck stiffness as opposed to the spinal rigidity is relatively more pronounced.

It is essential that lumbar puncture be done early in all these cases presenting meningeal signs since the cerebro-spinal fluid may provide the clue earlier than does the clinical examination.

Encephalitis. Sporadic cases of the various forms of encephalitis are always difficult to recognise in the acute stage. During an epidemic of poliomyelitis this difficulty is accentuated since encephalitis may initially resemble poliomyelitis to a considerable degree, and at such times it is reasonable to presume every case of encephalitis to be one of polio-encephalitis, unless there is good evidence against this diagnosis. At times it may only be possible to differentiate the one illness from the other by observation over a period, as well as by examination of the cerebro-spinal fluid on more than one occasion.

During the past year or so we have experienced on the Witwatersrand a number of cases of a type of meningo-encephalitis, possibly due to a virus now prevalent which is causing, *inter alia*, pneumonitis, gastro-intestinal disorders, hepatitis and adenitis. The encephalitis is difficult to differentiate from non-paralytic or encephalitic poliomyelitis clinically but examination of the C.S.F. would appear to reveal certain specific features, namely: pleocytosis in the C.S.F. with the polymorphs remaining present for longer than in poliomyelitis, high sugar and extraordinarily enough, high chlorides, the cause for which has not yet been satisfactorily determined. An initially raised protein in the C.S.F. falls rapidly with the disappearance of the cells, unlike in poliomyelitis where the protein actually rises as the C.S.F. cell count falls.

Peripheral Neuritis or 'Neuropathy'. Simple involvement of one peripheral nerve or massive involvement of all the large branches of a large nerve may occur in this neuritis, yet without any sensory changes and therefore will be very difficult to differentiate from poliomyelitis. The C.S.F. is usually normal in this type of neuropathy.

More extensive peripheral neuritis such as that consequent upon diphtheria or that of the Guillain-Barré syndrome is usually quite symmetrical, a state which seldom occurs in poliomyelitis unless the paralysis is extensive and complete. Further, the C.S.F. is normal in post-diphtheria polyneuritis and though abnormal in the Guillain-Barré syndrome it differs from poliomyelitis in that it exhibits a characteristic cell-protein dissociation. In these neuritides the prognosis is so very much better that it is well to be aware of the possibility of their occurrence.

The protean manifestations of poliomyelitis make the differentiation from many other illnesses necessary at times and passing mention need merely be made of some of these ailments, all of which were confused at one time or other with poliomyelitis in cases actually experienced in the two epidemics already referred to:

a. Tick-bite fever and glandular fever because of associated neurological manifestations, particularly headache, pain in the neck and back.

b. Osteomyelitis because of pain and immobility of a limb.

c. Typhoid fever owing to an acute onset with a number of the prodromal signs resembling poliomyelitis and a well-marked stiffness of the neck and spine.

d. Acute abdomen because of pain and urinary retention.

e. Falls resulting in trauma of a limb and consequent immobility. Beware in these cases of the actual trauma predisposing to the onset of an eventual poliomyelitis.

f. Hysteria.

Treatment. As yet we have no satisfactory specific treatment against the offending virus. Convalescent serum, even in the pre-paralytic stage, has proved disappointing in properly-controlled experiments and has now become almost entirely discarded. This is not really surprising when we remember that the virus probably travels to the cord and brain along nerve axons and is thus effectively protected against the action of antibodies.

Our treatment must therefore be along general symptomatic lines. During the expectant stage (meningeal phase) our attention will be directed chiefly toward determining the correctness of the diagnosis as also the localisation and gravity of symptoms. Where there is endemic poliomyelitis or certainly during epidemics, we would be well advised to advocate prophylactic rest and precautionary measures, should suggestive but non-specific signs and symptoms be present, however mild.

If the patient passes into an acute stage our treatment will be largely symptomatic and at times possibly life-saving. Our first duty, if the age permits co-operation, is to comfort and reassure the patient, attempting to secure relaxation. Undoubtedly the emotional state

and tensed attitude manifest in this disease exacerbate the symptoms, especially when bulbar and respiratory difficulties are present and we must seek to allay our patient's nervousness. The harmful influence of panic, fear and hysteria is often insufficiently appreciated.

Our next measure will be to determine whether the patient's life is in danger. Most deaths in the acute stage of poliomyelitis are due to failure of the cardiac or respiratory centres in the medulla or to disability of the respiratory mechanism. Diagnosis and treatment are facilitated if this respiratory mechanism is conceived in terms of its three main components, namely:

1. The respiratory centre.
2. The air passages with an important lock or potential trap in the pharynx and larynx under control of the lower bulbar nuclei.
3. The physical mechanism of the intercostal muscles and diaphragm controlled by the upper cervical segment.

A failing cardiac centre results in a rapid, irregular, thready pulse with a falling blood pressure. Affection of the respiratory centre renders the breathing shallow and irregular in rate and depth, with periods of apnoea and signs of anoxia and is of grave significance.

Lower bulb nuclear damage would be indicated by weakness of the soft palate and tongue with difficulty in swallowing, nasal regurgitation, nasal blurred speech, hoarseness and loss of gag reflex, inability to cough, pooling of secretions, stridor, dyspnoea and cyanosis.

Spasm of the intercostals and the diaphragm would result in interference with expansion and contraction of the thoracic cage and, in fact, if severe may reveal an almost immobile fixed chest. Paralysis of these muscles will mean insufficient power to inhale and exhale. We must particularly study the function of the diaphragm. One half may be immobile due to spasm, the other half, due to palsy, flapping up and down in the thorax and of little use to those muscles of respiration which are still functioning.

Involvement of the muscles of the anterior abdominal wall would contribute still further to decreased respiratory efficiency.

Treatment of all these will therefore depend on which factors predominate and careful watch must be kept for any manifestations of bulbar or spinal respiratory involvement.

The accumulations of mucus must be removed from the pharynx. The foot of the bed is raised to 30° to secure postural drainage, the patient being rolled onto his side or his abdomen and his feet being secured to prevent slipping. Repeated gentle aspiration is often required and at times when the excretions are copious it is necessary to employ suction through a laryngoscope or a bronchoscope. Atropin is contra-indicated as it renders the mucus too tenacious and difficult to expel.

The early use of moistened oxygen helps to support the bulbar centres and is best given through soft rubber nasal tubing, the flow usually being at about 4-5 litres per minute, in order to prevent cyanosis. Tents and masks are not usually practicable as they interfere with the attendant's efforts to aspirate the mucus.

Bulbar stimulation can be attempted, where indicated, by the use of appropriate drugs. Of these, caffein is the one most favoured. Falling blood pressure can be countered sometimes by the injection of epinephrin, neosynephrin or similar drugs.

Difficulty in swallowing necessitates parenteral feeding as there is great danger that food or fluid will be aspirated. Intravenous hypertonic 10% glucose may be given in the early stages in the hope of reducing central nervous system oedema but this must be shortly thereafter changed to an isotonic solution and care must be exercised that this be not given excessively, lest the salivary excretion become too copious. Should the swallowing still be difficult after several days, careful attempts can be made to pass a gastric tube, usually through a nostril this being left *in situ* during the day for introducing appropriate fluid feeds, vitamins, etc., but being withdrawn at night to permit more comfort during sleep. If introducing the tube evokes gagging and threats of vomiting the procedure should be abandoned. As swallowing becomes gradually possible small quantities of fluids can be cautiously fed to the patient, but care must be taken that the feed is not such that it would stick with the mucus in the throat. Incidentally milk is very inclined to do this and should be sparingly used at first.

Difficulty in respiration will call for further special measures. The progressive clinical stages of anoxia which warn us of impending respiratory failure are as follows: At first our patient seems unduly apprehensive, becomes restless and has difficulty in sleeping. As the respiratory effort becomes increased there is a disinclination to converse, sentences becoming more and more clipped. Twitching commences at the corners of the mouth, cyanosis becomes manifest and a comatose state gradually ensues.

Should the medullary respiratory centre alone be affected the respirator (iron lung) would be theoretically of assistance, but unfortunately in practice is found seldom to be of avail probably because of spread to the cardiac centres.

Bulbar patients with a good deal of pharyngeal mucus do badly in respirators as the airway to the lungs is partly obstructed; inhalation of mucus is bound to occur. If the measures already outlined for bulbar cases do not suffice to ward off anoxia, a tracheotomy under local anaesthetia must be undertaken to provide an entry for oxygen into the lungs and by-pass the pharyngeal and tracheal mucus. The trachea can be kept clean by suction and a valvular apparatus permits the delivery of the oxygen during aspiration. If the respiratory centre is involved as well, or if there is additional spinal respiratory palsy it might be essential to employ a respirator. In such case a preliminary tracheotomy would be essential. Bulbar cases may require antibiotics such as penicillin to ward off infection due to aspiration.

It is in the uncomplicated spinal respiratory palsies that the respirator is pre-eminently of value. One must first ensure that the difficulty in breathing is not merely due to spasm of the respiratory muscles. Relief of this spasm can sometimes be occasioned by the judicious application of hot packs to the chest and abdomen with consequent increased respiratory efficiency. If there is

actual severe paralysis of the muscles artificial respiration may be essential, and life can only be maintained by continued use of the respirator until there has been sufficient recovery of the muscles concerned to enable efficient spontaneous respiration to take place.

Certain cardinal points in regard to the use of the respirator can well be stressed:

1. Do not delay until the patient is in a dangerous anoxic state when irreparable damage may have become sustained by nerve cells.

2. Equally do not rush to use the apparatus unnecessarily. Patients may become psychologically dependent on this form of artificial respiration and they easily develop incorrect patterns of breathing which are troublesome to rectify later. Stimson has observed that there is a greater tendency to atelectasis in respirator patients. When in the respirator it is technically difficult to employ other measures of treatment which may be indicated, such as application of packs.

3. Wean as early as possible, even if only gradually. Sometimes intermittent use suffices.

4. Negative pressure should be used but avoid dangerously high pressure. Too great a negative pressure easily injures the alveoli in children. Children under 10 require about 10 c.mm. of water pressure and not over 14 c.mm. The rate is not so important as the height of the negative pressure. The patient rapidly adapts himself to the rate—20 to 30 per minute for most children.

5. Remember to maintain adequate nursing care while in the respirator and also to prepare the patient psychologically for the reception of this initially terrifying experience.

If satisfied that there is no imminent danger from bulbar and respiratory difficulties the following line of treatment can be pursued.

No matter how mild the case, even if considered an abortive one, adequate bed rest is advisable. One might here stress, in addition, the necessity for avoiding excessive activity and exposure to anything exhausting for at least three weeks after the abatement of temperature in all these abortive and mild cases. If there are more definite indications of central nervous system involvement special measures will have to be undertaken.

If there is appreciable dehydration due to vomiting or anorexia this should receive prompt attention and it may be necessary to give appropriate solutions intravenously.

The patient is put to bed on a firm mattress with a bed-board beneath it to prevent sagging. He is clad in a minimum of clothing and a cradle is provided to support the bed clothes and keep the weight of these off the patient. During the initial few days we must permit him to adopt whatever posture he prefers so as to give him maximum relaxation. Later he is encouraged to lie flat on his back without a pillow for his head and with his feet against a foot-board, unless there is spasm of the calf. He should be turned in bed several times daily, but care must be exercised to handle him gently.

Our primary consideration must be to make the child as comfortable as possible, without sedation, and yet to prevent or minimise possible deformities. During the early stages limbs should not be forced out of deformity but we should use gentle passive manoeuvres to reduce any bad positional attitudes. There is no indication for splinting in this phase but limbs may be kept in neutral positions by the use of light bolsters and pads. Sand bags are usually too heavy, but we found sawdust-filled bolsters useful.

Where there is a good deal of muscle tightness, small pads or pillows, judiciously applied would be helpful. For example: if hamstring spasm exists place a pillow beneath the knees when supine or beneath the shins when prone. For lordosis with tightness of the quadriceps place a pillow under the lumbar spine.

We must relieve muscle spasm and strain, give support and minimise pain as the latter is partly responsible for the patient's increased muscle tension. To maintain the muscles in as good condition as possible we must attempt to keep the involved areas well vascularised. In an endeavour to secure these requirements several measures may be undertaken.

1. Application of dry heat by the use of cradles fitted with electric bulbs. These are switched on for short periods of 20-30 minutes at varying intervals according to the requirements of the particular case. We employed this method extensively in the 1945 epidemic and found that it alleviated pain in a moderate number of instances.

2. Application of moist heat in the form of hot packs. It is not here intended to discuss the pros and cons of the physiological concept for the application of packs as put forward by Miss Kenny. All credit to her, at least for focussing attention on the management of certain specific findings occurring in the acute stage, namely muscle tightness or spasm, which had not been sufficiently recognised previously.

Suffice it to say that we found these hot packs most comforting in many cases and we employed them as extensively as our limited nursing facilities would permit. Space does not here permit discussion of the full technique, but one might add that we used laid-on packs applied to the affected parts for about 15-20 minutes, at as short intervals as practicable for several periods spaced over the day.

3. Paravertebral block: This method was used by Dr. S. N. Javett and the writer on several selected cases in the 1945 epidemic. We felt on theoretical grounds that this might relax spasm and at the same time provide warmth to the affected limb by securing vaso-dilatation. We were able to relieve spasm promptly but in most cases only transiently and therefore regarded this method merely as a pointer to some other possible future approach.

4. Our own experience in the 1945 epidemic led us to believe that spasm might be a manifestation of sympathetic over activity and in last year's epidemic we considered it worth trying a drug which would block the transmission of impulses through autonomic ganglia. To this end it was thought feasible to use tetra-ethyl-ammonium chloride (Etamon. P.D. & Co.) intravenously and a preliminary investigation was undertaken by Mr. J. Lannon and Dr. J. L. Braudo. Their report appeared in the South African Medical Journal earlier this year and indicates that this method can relieve spasm, if only for a period, does decrease pain and brings warmth to

cold clammy limbs. If this procedure can thus improve the mobility of affected parts in those cases rendered immobile by spasm rather than paralysis it will contribute towards minimising deformity.

Lest it be considered that this matter of spasm is being unduly laboured we must stress our belief that muscle tightness plays a very important role in producing deformities in poliomyelitis. We feel it essential to take measures against this spasm when it commences in the early phases of the disease.

When the sensitivity and irritation of muscles is reduced we can passively develop ranges of normal movement, gradually increasing the arcs of motion in the affected area, but details of this and of later active exercises will be given by those dealing with the treatment in the convalescent stage.

Retention of urine may cause a good deal of discomfort and may be severe enough to require catheterisation. If the difficulty persists for more than a couple of days it may be wise to fix in a self-retaining catheter and to give appropriate oral chemo-therapy for urinary antisepsis. Recently injections of furmethide have been advocated but we have had no experience with this. Constipation may require daily enemata.

Various other drugs have been advocated for the treatment of poliomyelitis. Of these only two need be mentioned here, namely, neostigmine and curare. The former lowers the barrier at the motor end-plates and should facilitate the passage of stimuli from the C.N.S. to the muscles. We have tried this preparation extensively but cannot confidently state it to have been of any real value.

Curare and allied preparations are now evoking great interest. These act by raising the barrier to nerve stimuli at the motor end-plates and so would shut off some of the motor impulses that are increasing muscle tension and thus should relax spasm. This is effectively done but the margin of safety is narrow and until the method is better standardised it could scarcely be put to general use.

It must be understood that as yet we have no specific therapy for poliomyelitis. At best we assist in the regeneration of neurons partially affected by the virus or we help to keep the involved muscles in a better state. Our treatment is of a supportive nature designed to restore to the patient the maximal physiological stability it will be possible to secure. The real answer to the problem of poliomyelitis will be the discovery of specific immunising therapy.

POLIOMYELITIS

POST-PARALYTIC TREATMENT — ORTHOPAEDIC ASPECT*

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The fact that the treatment of poliomyelitis has devolved gradually on the orthopaedic surgeon is an indication of the failure of any therapeutic agent to alter the course of the disease. Seddon has stated that there is no specific treatment and recovery depends upon the spontaneous return of function to such of the anterior horn cells which have not been completely destroyed. No one, up to the present time, has succeeded in curing a case of poliomyelitis. Every one familiar with the disease must be struck by the different rates of recovery of patients receiving precisely the same type of treatment. Some cases show no recovery at all. It must be understood clearly that treatment of the limbs has no specific effect on the course of the disease in the spinal cord, and, that, on the recovery of the anterior horn cells depends the outcome of the disease. It may be suggested that no treatment is of any value. This is not true, however, for placing the limbs in the best position for recovery is of the utmost importance. Permanent paralysis is minimised by the prevention of contractures. Constructive surgery will be needed at a later date to repair in part, the ravages of the disease. It is in this dual role that the function of the orthopaedic surgeon lies.

* This paper was read at the Medical Congress of the Association held at Cape Town in September 1949.

The first problem of the surgeon is, therefore, to place the affected portions of the body in the best possible position for recovery. Although recovery, so far, is outside medical control, at least it should not be hindered by allowing contractures to develop.

It cannot be too highly emphasised that the prevention of contractures requires the services of a skilled and trained staff, both nursing and medical. Deformities have an insidious habit of developing, as is only too well realised by those who have attempted to control their onset in cases of rheumatoid arthritis.

The second problem is, where recovery has not occurred, to deal with the residual paralysis and to give the patient the best possible use of the limbs. It has been stated that recovery takes place up to two years from the onset of the illness. In actual practice, significant recovery takes place in the first six months, and, although some improvement does take place after this, it is not marked. Exceptional cases do occur where there is recovery after six months but they are unfortunately rare.

DIAGNOSIS

In comparatively recent years poliomyelitis has been recognised as highly infectious so that the first three weeks are spent in an isolation hospital. As a rule, cases

are referred to the surgeon after this period, that is, at a time when the diagnosis is established. The characteristic asymmetrical paralysis without loss of sensation, is not easy to mistake.

Old cases, in addition to the paralysis, have a poor circulation which may cause chilblains and shortening of a limb, due to a slowing up of growth at the metaphysis.

PATHOLOGY

The details of pathology will have been discussed by others. There are, however, certain points worth mentioning. It has never been clear to the clinician why there should be pain, tenderness and spasm associated with paralysis of muscles. There is evidence to show that the inflammation is widespread in the central nervous system. Anderson states, that while there is unquestioned predilection for the anterior horn cells, few inspections fail to reveal evidences of microscopic disarray throughout the nervous system.

There appears to be general agreement that the usual case presents maximum involvement early. And the supposition is that the virulence of the virus has spent itself within a matter of days. There are, however, often continued spasms and tenderness for a much longer period. Kabet and Knapp suggest that these phenomena are the result of damage to the internuncial fibres during the acute attack.

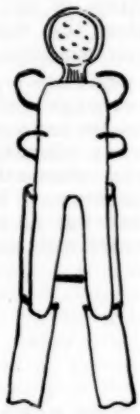
Howe and Bodian state that some encephalitis, although not clinically evident, is a constant feature of poliomyelitis. Later Bodian, in discussing this question, believed that the spasm is more likely to be due to cerebral damage. This certainly would be more in accord with experience in regard to other forms of spasticity in muscles. There are many aspects of poliomyelitis which are still not fully understood.

Lannon and Braudo have been able to abolish pain and spasm in poliomyelitis by using a drug called Etamon which temporarily blocks the transmission of impulses through the autonomic ganglia. They infer from this that the spasm is due to the overactivity of the sympathetic. This is a most interesting observation in view of the work of Royle and Hunter more than 20 years ago, in which they demonstrated dual innervation of muscles and claimed that the sympathetic fibres controlled tone and spasm. For spastic paralysis they devised the operative removal of the sympathetic ganglia. These operations were a failure and did not alter the spasm of muscles in spastic paralysis. Other effects were noted, such as the relief of spasm of the colon and increased blood supply to the limbs and as a consequence Sympathectomy has become a common operation for vascular conditions.

Lannon and Braudo's work appears to show that, after all, the sympathetic does play some part in the production of spasm in muscles and they have investigated a drug which may prove to be a most useful therapeutic agent.

PREVENTION OF DEFORMITY DURING ACUTE AND CONVALESCENT STAGES

As has been stated, the first three weeks of the disease are spent in an isolation hospital, and it is most desirable that treatment should begin during this stage. The handling of the acute phase of the disease should be of major importance to the surgeon who has to handle the late results, since his task may be much simplified and his results much better if the optimum treatment is accorded to the patient from the beginning. A certain amount of difficulty arises. Is it better to treat the case in an orthopaedic hospital where facilities for isolation are poor or in an isolation hospital where facilities for treatment are poor? Seddon states that on the whole the latter is safer but that orthopaedic staff should be attached to the isolation hospital so that the cases can be under supervision and treatment from the earliest possible moment.



Jones frame

Treatment in the early stages of medicine has aroused considerable controversy as to whether splints should be used to prevent contractures or not. In general, it can be said that the more skilful the nursing and physiotherapy staff in posturing the limbs, the less splints are necessary. However, it is the opinion of the majority of orthopaedic surgeons that well-fitting, removable plaster of Paris splints are useful for the relief of pain, also for application at night to prevent deformity. During the day it is less necessary to use any splints and the majority of cases can be postured with pillows, sand bags and bed boards. There is always the difficult child with marked muscular imbalance of the spinal or abdominal muscles who will need to be controlled on a frame or a plaster bed. For example, paralysis of the left spinal or abdominal muscles with paralysis of the right glutei will tilt the pelvis and produce a scoliosis and even dislocation or subluxation of the right hip joint.

The keenest antagonist of the case for splints is Sister Kenny and as her methods have received a great deal of publicity, both in medical and lay press, she deserves mention.

Sister Kenny postulates three factors which distinguish her concept of poliomyelitis. They are somewhat difficult to understand and seem to bear no relation to normal pathological or physiological terms. The first is 'muscle spasm' which occurs in the opposing muscles and prevents contraction of weakened muscles. The second is

'inco-ordination and dissociation', consisting of contractions or delegation of function by one muscle to another. The third is 'mental alienation' or inability to contract a muscle voluntarily. The treatment given is hot packs or fomentations to relieve spasm, and muscle re-education to prevent mental alienation.

No splints, braces or corsets are used while under treatment, although some of these measures may be necessary after Sister Kenny has finished her treatment.

If a case recovers it is claimed as a cure for the method, no cognisance is taken of the possibility of spontaneous recovery. Sister Kenny's methods have been thoroughly investigated, both in Britain and the United States. A committee to investigate her treatment in Britain was not impressed and stated that her methods were efficient but the final results were no better than more orthodox treatment. In America, Krusen reported favourably on her treatment; later, however, Gill, Key and Rechtman independently saw no advantage in her methods and some disadvantages. The Kenny methods are very expensive and require the patients to be left recumbent for a period up to two years, and when her treatment is discontinued contractures may still occur which require braces and splints.

In America a committee consisting of Ghormley and his colleagues investigated 740 patients with poliomyelitis, 650 of whom had been treated by the Kenny method. This committee concedes that the publicity had brought about renewed interest in certain principles of treatment described by Robert Jones in 1911 and Lovett in 1917 and since claimed as original by Sister Kenny. (Stretch paralysis and functional loss of use by Jones, and moist heat, muscle training and pools by Lovett.)

On the whole the Kenny system may be summed up as an intensive course of physiotherapy consisting of the application of hot fomentations or hot packs, massage, muscle re-education and muscle training, without the use of splints. The picture is not encouraging nor does it give rise to the hope that in the so-called 'Kenny system' lies a reasonable solution of the prevention of paralysis.

There are certain maxims for guidance during the acute and convalescent stages.

1. All affected or paralysed muscles to be held in the relaxed position. During the acute stage and while there is any temperature, pain or tenderness, the affected limbs and muscles should be kept as quiet as possible. The application of splints is indicated at this stage, to hold each affected muscle in a relaxed position. Heat, either wet or dry, is useful for the relief of pain and spasm. Well-fitting splints for feet and legs can be made from plaster of Paris and should be worn continuously while there is pain and tenderness.

Other practical suggestions for the management of paralysis are:

Shoulder: Abduction. The upper arm is placed within a roller towel and the top of the roller towel tucked into the mattress at the top of the bed. A soft, thin pillow can be placed under the point of the elbow to prevent strain on the anterior part of the capsule of the shoulder joint. Rotation of the shoulder joint is maintained in mid-position by sand bags or allowed to spend equal times in extreme positions.

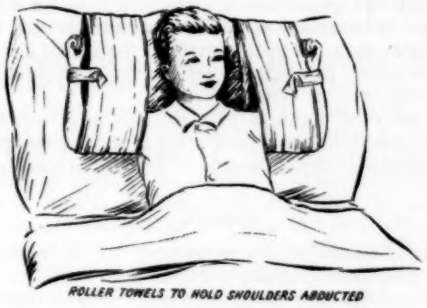


Fig. 1.

Elbow. Mid-position can be maintained with sand bags or by pinning the shirt sleeve to the roller towel.

Fingers can be bandaged over table-tennis ball or larger ball according to the age of the patient.

Back. Lying flat on a firm mattress. A small, flat, felt pad may be placed in the lumbar region.

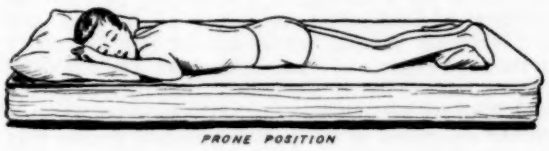


Fig. 2.

Hips. Flat with legs slightly apart. A long sausage shaped sand bag can be placed on either side of both legs to prevent their rolling in or out and also to keep them apart.

Knees. A small pillow or rolled soft pad can be placed under the knees to keep them flexed about 10 degrees.

Ankles. Firm, right-angled sand bag should be placed at the soles to keep the ankles at right angles or a padded, wooden box with weights inside it, can be placed against the feet.

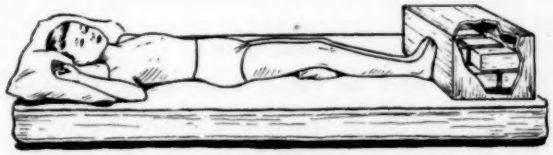


Fig. 3.

IRON LUNG

Iron lungs in this centre have remained under the control of the Isolation Hospital, because the lungs are a necessity in the first few days of the disease. The staff have become adept in the use of it, and have overcome the difficulties in the technique of managing a patient in the iron lung. Lives are saved with this apparatus, but it is a mixed blessing. The plight of the patient, whose

respiratory muscles do not recover and who cannot live without the iron lung is familiar to us all.

2. No physiotherapy should be given until all pain, tenderness and spasm have disappeared from the muscles. Massage or stimulation while the muscles are tender only causes pain. Painful stimuli to the spinal cord will, for a certainty, not help to relieve the inflammation in the central nervous system.

The work of Lannon and Braudo has shown that Etamon injections can relieve and cut shorter this painful stage and allow physiotherapy to be commenced sooner.

CURARE

Ransohoff described the treatment of 29 cases with curare. The claims made, are that pain and spasm are relieved and the results are improved. Seddon states that it is very doubtful if it is completely effective in relieving painful muscle disorders.

Curare may have some effect in relieving discomfort, but it is difficult to see how it can in any way alter the course of the disease.

HYDROTHERAPY

At the risk of repetition one point requires stressing, and that is the advantage of a swimming pool for the treatment of paralysis as opposed to a large bath such as a Hubbard. Hospital authorities are loth to embark on the extra expense of a swimming pool. The difference is comparable to having a bath and going for a swim in a pool. Both are wet and both can be the same temperature but the enjoyment of the pool is infinitely greater than the bath. It is exactly the same in poliomyelitis. Several children can be in the bath at the same time, games can be organised and supervised by the physiotherapist. The whole atmosphere is one of intense enjoyment which is good for morale and good for treatment. The Hubbard bath can only deal with one patient at a time, so that in the event of an epidemic, it means that each patient gets to the bath only once or twice per week for short periods. In large centres where poliomyelitis is treated in considerable numbers a small swimming pool is an essential part of treatment. It is the accepted practice in Europe and America and yet there is only a single pool for this purpose in the Union of South Africa.

CHRONIC STAGE

This begins when no further improvement is taking place, which may be a period of about six to eight months to two years.

Bradley and Gale give figures for the 1947 epidemic in England and Wales; 7,500 cases were notified and information was obtained about 4,717 cases admitted to hospitals. Of these 63% had paralysis of the limbs or trunk, 10.3% had paralysis of the cranial nerves and 26.7% had no paralysis. Of the 3,461 paralytic cases, 333 were fatal, and of the remainder 38.5% were classed as moderate and 20.4% as severe. It was, therefore, estimated that 3,000 patients would need long term treatment in hospital with about 33% under the age of five years. The severely crippled would number about 1,000. These figures serve to show the extent of the ravages of the disease and the fact that in England and Wales there were 1,000 severely paralysed cases and between 1,500 and 2,000 moderate cases who would need surgery to repair or minimise their disabilities.

The disease still affects mostly children and in the above figures 33% were under five years of age. Although there is a marked increase in the number of adults affected in recent epidemics, the main problem remains one of paralysis during childhood. This, in itself, brings in special complications in so far as surgery is concerned, because deformity and distortion of the limb or trunk are liable to occur or increase during the period of growth. Constant supervision of each child with a paralysis is necessary during childhood and not until after puberty, 14-16 years, is it safe to perform operations on bones or joints which will finally stabilise the deformity. Adequate supervision is a difficult problem in South Africa, on account of the long distances involved, and made still more difficult by the fact that there are different authorities controlling hospitals and health services outside hospitals. After-care clinics have been established, after a hard struggle, in the Cape Peninsula to supervise cripples in general, as the problem during the growth of the child is the same for all deformities. These clinics have been a tremendous success. They have provided treatment for cases living too far from a hospital, minimised the length of stay in hospital and helped to prevent recurrence of deformities. The consequence is a much more efficient use of hospital beds. The system of clinics is simple and inexpensive. A similar system spread over the country is badly needed for all cripples of whom poliomyelitis cases form a not inconsiderable part.

While up to the present there is no means of preventing paralysis, curing it or even controlling an epidemic, a great deal can be done to help those suffering from the disease. No system of hospitals can provide continuous supervision for more than a few who live nearby; in fact, hospitals are no more equipped to deal with cripples than they are to deal with tuberculosis or venereal disease. Under the amended Health Act a subsidy of 100% is provided by the Government to a Province or hospital board for the establishment of an out-patients' clinic at a distance from a hospital. For a local authority the subsidy is 50%. This subsidy is for services at the clinic only and does not include any home visiting. It is difficult to understand why 50% is granted to a local authority as opposed to 100% to a Province or hospital board. The fact that there is a subsidy is a strong encouragement to other authorities to develop this essential after-care work. The National Council for the Care of Cripples is giving all help possible to this project and has granted funds which, with the help of the local authorities, has enabled after-care and out-patients' clinics to be established in the Peninsula.

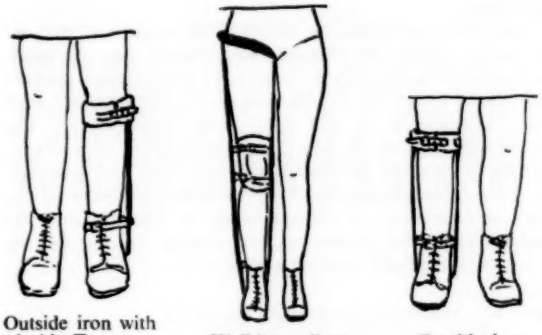
Stress has been laid on the development of deformities in children while they are growing. The surgeons' main objective is to prevent deformity by the simplest possible means, until a final correction at 15-16 years can be performed. As practically every conceivable deformity can occur the treatment entails diverse and numerous methods of which only a short summary can be attempted here.

There are three methods at the disposal of the surgeon:

1. Irons, calipers, corsets and splints.
2. Manipulations and tenotomies followed by immobilisation in a plaster of Paris cast.
3. Operations, which for children, are mainly on soft tissues and for adults are mainly on bones.

APPLIANCES

An appliance is an apparatus for assisting locomotion and preventing deformity. It is an unfortunate fact that the greater the handicap the heavier and more elaborate the appliance which must be used. There are many different types of appliances—only the most useful ones will be mentioned.



Outside iron with inside T strap | Walking caliper | Double iron

Outside iron with inside T strap is used to control overaction of the peronei.

Inside irons with outside T straps are used to control overaction of the invertors of the foot, that is, tibialis anterior and posterior.

In association with these irons may be used a *spring* for drop-foot or foot stops to prevent either an equinus or calcaneous deformity.

Double iron below knee is used to give stability to the foot where there is almost complete paralysis and may be combined with stops to prevent either equinus or calcaneous deformities.

Walking caliper is necessary where there is paralysis of the quadriceps or hamstrings causing instability of the knee joint.

Walking caliper and waist band are necessary where two calipers must be worn, or where there is paralysis of the hip muscles causing an external or internal rotation deformity at the hip joint.

Abdominal belts are necessary where there is paralysis of the abdominal muscles. If this paralysis is symmetrical it can be controlled by an abdominal belt, sacro-iliac belt or an elastic belt. Scoliosis will develop where there is asymmetrical paralysis of either the abdominal or spinal muscles. Scoliosis, under these circumstances is almost impossible to prevent and very difficult to control and, as a result, there are numerous types of appliances and corsets. One of the best types has been developed in the childrens' hospital in Boston, U.S.A. It has two adjustable arms which hold and support the convexities of the scoliosis. There are no efficient appliances for arms and hands. Splints are used to prevent deformities and abduction splints for the shoulder and a cock-up splint for the wrist.

MANIPULATIONS AND TENOTOMIES

The deformities of poliomyelitis are mainly of the soft tissues. The capsule and muscles are shortened on one side of a joint and lengthened on the other. These deformities can be overcome with continuous traction where this is applicable, i.e., to knee and hip. With feet, however, continuous traction is difficult and impracticable to apply. The method of choice is repeated manipulations and plaster of Paris. If a deformity is not overcome at the first attempt it is amazing how the tissues will soften in a plaster of Paris cast, thus allowing correction to be completed at the second or third attempts. Where there are strong tendinous structures such as a contracted tensor fasciae femoris, plantar fascia or a contracted tendo Achillis, causing the deformity, a subcutaneous tenotomy of these structures with a small-bladed tenotomy knife will facilitate correction.

Care has to be taken in carrying out a tenotomy of the tendo Achillis: the tendon must not be cut across in one place, but must be partially divided on opposite sides—the two cuts must be at least 1 inch to 1½ inches apart. This gives a Z-shaped cut enabling the tendon to unite again firmly.

The disadvantage of manipulative correction is that the conditions which produced the deformity in the first place, are still present, so that recurrence or relapse may occur. Muscle balance may be restored by a suitable tendon transplantation, or, if the patient is old enough, by an arthrodesis. In many cases, however, conditions are such that there is no suitable tendon transplantation, and the patient has not reached a suitable age for an arthrodesis, 14-16 years. In such circumstances it is necessary to persist with manipulations, and, if available, physiotherapy. When a relapse takes place the manipulations can be repeated.

OPERATIONS

The operations which may be performed for poliomyelitis are many and diverse. The generally accepted types of operations with their indications and objectives will be enumerated. It is not the function of this paper to give a complete list of all the operations used in the treatment of poliomyelitis.

Operations on Soft Tissues may be carried out at any stage; although the older the child is, the less risk there is of relapse, and when very young, as in infants, a tenotomy may serve the purpose quite as well. This fact is of mportance and will be mentioned with the description of any particular operation.

Muscle Transplantation. This comprises a group of operations which was in considerable vogue 25-30 years ago and in which practically every muscle in the body had been transplanted. Through the process of trial and error a great many of these have been discarded and a few only have stood the test of time.

These operations are indicated where there is marked

muscle imbalance, causing deformity. The operations can be performed at any age and have the effect of improving function and preventing deformity. A transplantation will not cure a deformity. The deformity must be corrected by either manipulation or other operative means before a transplantation will act. Particular transplantations which can be recommended are:

Transplantation of the tibialis anterior to the outer border of the foot on account of weakness or paralysis of the peronei, causing an inversion deformity of the foot. The tibialis anterior is detached close to its attachment in order to get as long a tendon as possible. It is passed through a tunnel in the tarsus or meta-tarsus and sewn to itself. The tibialis anterior is a strong muscle and experience teaches that it is not necessary to transplant further out than the midpoint of the foot, otherwise the opposite deformity of eversion may be produced. Judgment is required, as the precise site will depend on the strength of the tibialis anterior and the degree of weakness of the peronei. A correctly-transplanted tibialis anterior can give an excellently-functioning foot, without any deformity or the need for a supporting iron.

Transplantation of peroneus longus to the inner side of the foot. The peroneus longus is not a powerful invertor of the foot when transplanted. It does, however, serve to restore muscular balance and prevent an eversion or valgus deformity of the foot, caused by overaction of the peronei.

Transplantation of peronei, tibialis posterior and flexors of toes into the tendo Achillis. Paralysis of the tendo Achillis causes a severe calcaneo-cavus deformity of the foot; a most unsightly and incapacitating deformity. After correction of the deformity, transplantation of the acting plantar flexors into the tendo Achillis will improve function of the foot and help to prevent recurrence of the deformity.

Transplantation of flexors of the wrist joint into extensors of fingers and thumb. This is an excellent operation, although the precise indications are rare in poliomyelitis, that is, paralysis of the extensors of the fingers and wrist with acting flexors. Flexors carpi ulnaris and radialis are transplanted into the extensor tendons of the fingers and palmaris longus into the thumb extensor tendons.

Transplantation of palmaris longus or flexor carpi radialis or ulnaris into the base of the first metatarsal is useful to improve the function of the thumb in cases of paralysis of the small muscles of the hand, which is not an uncommon sequela of poliomyelitis.

Transplantations require four to six weeks of immobilisation following the transplant to allow the tendon to become fixed, after which active re-education of the transplanted muscle is carried out.

Pes cavus deformity of the foot may be caused by a paresis or weakness of the extensor muscles and does not develop when there is complete paralysis of the extensors, or as mentioned earlier, may develop with a calcaneus deformity, due to paralysis of the tendo Achillis. The arch of the foot acts like a bow with the soft tissues, i.e., plantar fascia, short flexor muscles and plantar ligaments, acting as the string of the bow. The arch of the foot cannot be flattened, unless the soft tissues are divided. Steindler described the operation which retains his name. It consists of detaching the plantar fascia and short muscles from the medial tuberosity of the os calcis, the plantar ligaments are divided and the foot is then manipulated flat with a Thomas' wrench. The foot is then encased in a plaster cast for six seeks. In cases of children, a subcutaneous tenotomy of the plantar fascia followed by wrenching is usually sufficient, but with adults or adolescents the full operation should be carried out and in some cases a wedge of bone has to be removed from the dorsum of the foot.

Flexion deformity of the hip joint is another very common condition. In children a tenotomy of the tensor fasciae femoris, followed by traction on the limb will be sufficient.

Where the deformity is firm and resistant and old, an operation called the 'Soutter operation' may be used. It consists of detaching the origins of the gluteus medius and iliacus from their attachments to the external and internal surfaces of the iliac bone respectively. On forcibly extending the hip joint the muscles slide away, leaving a portion of the iliac bone with the anterior superior spine projecting. The projecting portion of the ilium is removed. A plaster spica or traction may be applied after the operation, depending on the degree of correction which has been obtained.

CONTROL OF GROWTH

Shortening of a limb due to paralysis is one of the unfortunate sequels and the shortening is in proportion to the degree of paralysis and the age of onset. The surgical ideal is therefore to have limbs of equal length when growth is complete.

The following methods are used:

1. *Bone Lengthening*. This is a procedure fraught with considerable risk. The devitalised tissues of the paralysed limb are liable to necrose under tension. Sepsis is a possible complication as four pins are fixed in the bone for a long period. Lastly, non-union of the bone is very liable to occur, especially if the operation is attempted in adults. For these reasons careful consideration has to be given before the operation is attempted, and should only be carried out where experience and facilities are adequate.

The bone selected is the tibia and a successful result will give a lengthening of two inches. The tendo Achillis is lengthened, pins are inserted, two into the upper and two into the lower end of the tibia, then a long oblique osteotomy is performed. The pins are connected with a distraction apparatus which gradually lengthens the limb.

In adults non-union is very liable to occur and will necessitate a bone graft. The operation is therefore not one to be recommended for adults with any degree of assurance but may be more safely carried out in children from 8 to 12 years of age.

2. *Arrest of Growth of the Sound Limb*. Excision of the epiphysis on the sound limb at a suitable age will cause shortening.

The epiphyses at the lower end of the femur and upper end of the tibia are selected. The operation is simple but

the amount of shortening is difficult to predict as individuals vary considerably. Tables are available to indicate the expected shortening which will take place.

3. *Bone Shortening*. This requires a major operation on the sound limb. If any complications, as sepsis or non-union occurs, a catastrophe results. This operation is, therefore, not looked upon with great favour.

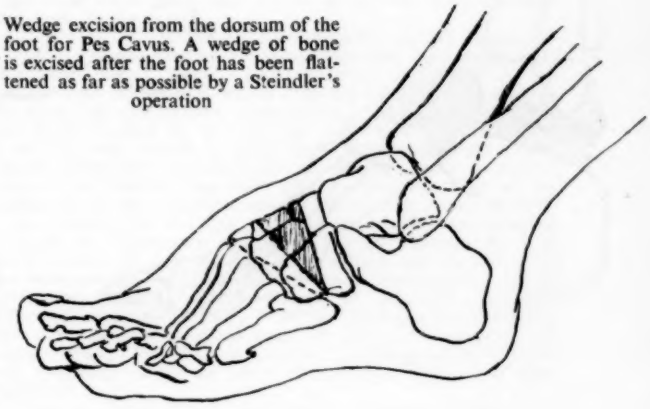
4. *Sympathectomy*. Harris and McDonald state that if this is performed on children the rate of growth is increased.

The results, however, are not consistent and more confirmation is required. The operation does hold out prospects for those cases of severe paralysis of a limb with its attendant poor blood circulation.

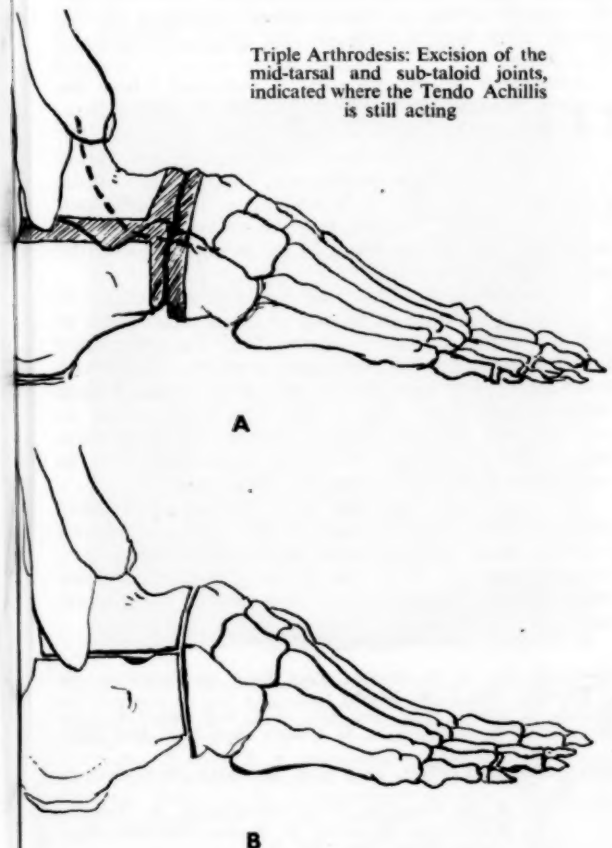
DEFORMITIES OF BONE

Operations which may be performed at any age:

Genu Valgum. Although knock knees may be controlled or corrected by the use of either knock-knee irons or knock-knee straps on a walking-caliper, an operation is sometimes necessary. The deformity is more frequently in the lower end of the femur but may be in the upper end of the tibia. An osteotomy of the lower end of the femur or a wedge osteotomy of the upper end of the tibia corrects the deformity. The bone unites readily in six to eight weeks, and there is little risk of any complication.



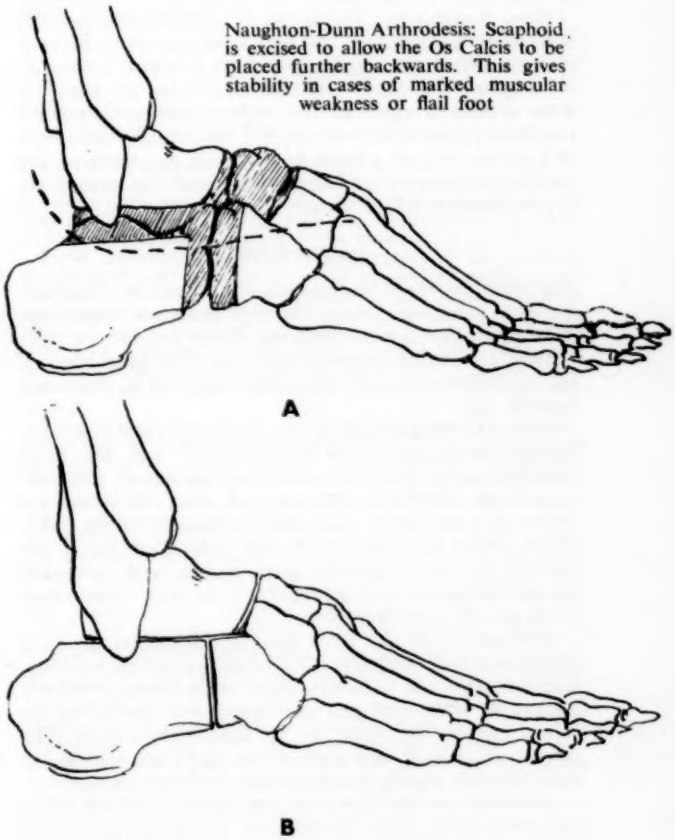
Wedge excision from the dorsum of the foot for Pes Cavus. A wedge of bone is excised after the foot has been flattened as far as possible by a Steindler's operation



Triple Arthrodesis: Excision of the mid-tarsal and sub-taloid joints, indicated where the Tendo Achillis is still acting

A

B

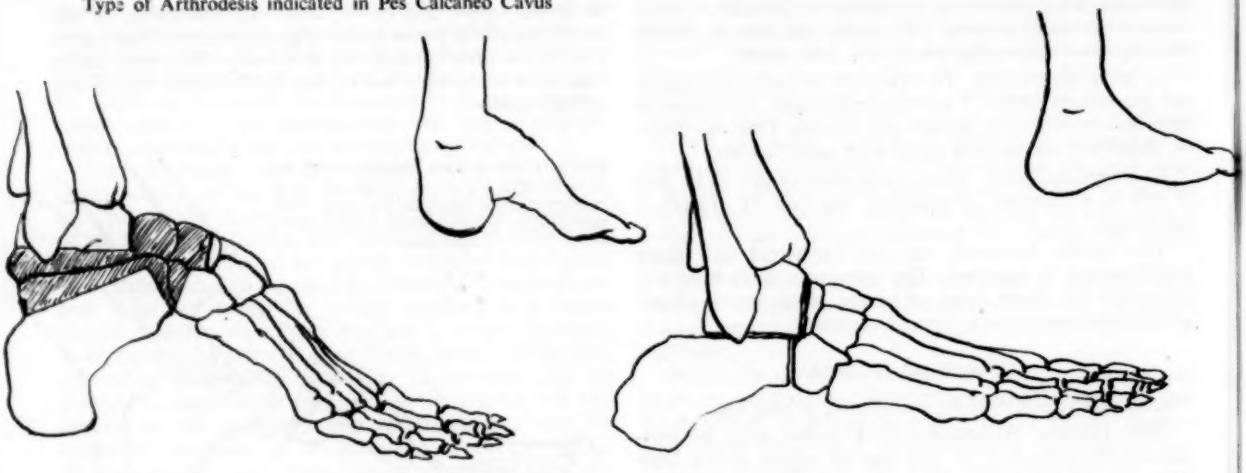


Naughton-Dunn Arthrodesis: Scaphoid is excised to allow the Os Calcis to be placed further backwards. This gives stability in cases of marked muscular weakness or flail foot

A

B

Type of Arthrodesis indicated in Pes Calcaneo Cavus



A B

Genu Varum. This is not a common deformity in poliomyelitis and can be corrected by either osteoclasis or osteotomy of the tibia.

Genu Recurvatum. This is due to a tight tendo Achillis with weak hamstrings. The best treatment is to prevent it or to relieve the conditions which produce it, that is, to lengthen the tendo Achilles and later see that the knee is held in slight flexion with a caliper. Operation is difficult; it entails either a special osteotomy of the upper end of the tibia or a bone block made by attaching the patella to the upper end of the tibia and thus presenting hyperextension of the knee.

ARTHRODESES

The most common operations performed for paralysis are arthrodeses of joints. For this group of operations it is advisable to wait until the bones are almost fully grown, as further growth-distortion may take place if the operations are performed too early: 14-16 years is a suitable age.

Paralytic Deformities of the Feet are very common. Several operations have been devised and the basic principles of each is to abolish movement at the mid-tarsal and sub-taloid joints in such a way as to correct deformity, and allow controlled movement at the ankle joint. When the mid-tarsal and sub-taloid joints are excised, the foot becomes more stable, and, provided the paralysis does not extend above the knee, supporting irons can be disposed of.

Arthrodesis of the Knee has the disadvantage of a permanent stiff knee which is a handicap not for walking, but for sitting. A walking-caliper with joints, enables a patient to walk with the knee rigid, but unlocking the joints enables the knee to be flexed for sitting. The operation is only indicated where there are strong hip muscles with paralysed quadriceps and hamstrings.

Arthrodesis of the Hip is only indicated in a limb where the knee of the same limb is well controlled by muscle power and the hip muscles are paralysed. It has two disadvantages. The first is inconvenience in sitting, and the second is that an added strain is thrown on the lumbar spine with consequent risk of osteo-arthritis in later years.

Arthrodesis of the Shoulder is indicated where the scapular muscles are strong in association with a paralysed deltoid. An excellent result can be obtained, but a careful study of the patient's work and habits should first be obtained. For example, after an arthrodesis of the shoulder it is difficult for a woman to do up her hair. While increased function may be obtained in other directions, the patient may not be so satisfied with the results as the surgeon.

Arthrodesis of the Spine. An extensive arthrodesis of the spine is required in certain of the severest types of scoliosis which occur in poliomyelitis. The spine must first be corrected as far as possible in plaster jackets with turnbuckles. The operation is carried out through large windows cut in the plaster cast. The operation is an extensive one and will have to be done in two or three stages. The Hibb's type of fusion of the spine may be carried out or the Albee type with bone grafts.

All in all, with suitable early treatment, adequate after-care and operative repair, a great deal can be done to help those afflicted with paralysis. Unfortunately it is only help and not a cure. Either prevention or some means of attacking the virus *in vivo* must be the constant aim.

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NEW PREPARATIONS AND APPLIANCES

STIGMINENE BROMIDE 'WARNER'

(1-BENZYL-3-(DIMETHYLCARBAMYLOXY)-PYRIDINIUM BROMIDE)

The effect of cholinergic or parasympathomimetic drugs is believed to be due to inhibition or inactivation of cholinesterase, thus resulting in the conservation or accumulation of acetylcholine in the tissues. With respect to the gastrointestinal tract and the urinary tract, the effect of cholinergic drugs is to improve the tone and motility of the smooth musculature.

The new cholinergic compound, Stigminene Bromide 'Warner', has been developed by the Warner Research Institute, to provide the surgeon with an improved means for the successful prevention or treatment of post-operative abdominal distension and urinary retention, the result of intestinal or urinary bladder atony.

Indications: Stigminene Bromide 'Warner' is indicated for the prevention and treatment of gastrointestinal and urinary bladder atony or post-operative abdominal distension and urinary retention.

Dosage: *Prevention of post-operative abdominal distension; or urinary retention; intestinal atony; urinary bladder atony*: One c.c. (0.5 mg.) by intramuscular injection immediately following conclusion of the operation. Repeat the intramuscular injection of 1 c.c. (0.5 mg.) every 3 to 4 hours until a total of 6 injections (including the injection immediately following the operation) has been given. If necessary, further injections (over and above the total of six) may be given at intervals of 4 hours.

Treatment of established post-operative abdominal distension: One c.c. (0.5 mg.) by intramuscular injection, followed by a small low enema 20 to 30 minutes after the injection. Repeat the intramuscular injection of 1 c.c. (0.5 mg.) every three hours until a total of not less than six doses has been administered.

Treatment of established post-operative urinary retention: One c.c. (0.5 mg.) by intramuscular injection, then apply heat (hot-water bottle, electric pad) to lower abdomen. If the patient does not urinate within 1 hour following the injection, he should be catheterized. Irrespective of whether the bladder has been emptied by spontaneous voiding or catheterization, 1 c.c. (0.5 mg.) should be given every three hours until a total of five doses (not including the first injection prior to emptying of the bladder) has been given. The first dose of the series of the five injections should be injected intramuscularly 2 to 3 hours after the time at which the bladder was emptied.

Caution: Stigminene Bromide 'Warner' should be used with caution in patients who have asthma, and should never be administered in cases of mechanical obstruction. In the event of excessive or over-dosage of Stigminene Bromide 'Warner', atropine sulphate, 1/100 to 1/150 grain administered by intramuscular injection may be used to counteract the drug's effect. This dose of atropine sulphate may be repeated as necessary until the desired antidotal effect has been obtained.

Package Information: Stigminene Bromide 'Warner' is available in 1 c.c. ampoules of a 1:2,000 solution (0.5 mg. each), cartons of 6 and 50 ampoules.

Distributor: William R. Warner & Co. (Pty.), Ltd., 6-10, Searle Street, Cape Town.

IN PASSING

INTERNATIONAL SERODIAGNOSTIC CONFERENCE (SYPHILIS) TO BE ORGANIZED BY THE WORLD HEALTH ORGANIZATION

International serodiagnostic conferences have been held by the League of Nations, 1923 and 1928 in Copenhagen, 1930 in Montevideo, and by the United States Public Health Service 1941 in Washington, D.C. In 1939 a similar conference planned in Copenhagen was cancelled at the outbreak of the war.

These conferences contributed significantly to the technical development and standardisation of serological methods and antigens in the diagnosis of syphilis. It has been recognized that conferences of this kind are the only means of comparing efficiency in test methods, of which as many as twenty different ones were compared at international serodiagnostic conferences in the past, at the same time offering a rare opportunity for exchange of scientific technical information between test-authors.

The Interim Commision of the WHO (1947) and its Advisory Committee on Venereal Diseases pointed out the necessity of holding further serodiagnostic standardisation conferences. The First (1948) and Second World Health Assemblies (1949) emphasised the need for further standardisation of serodiagnostic laboratory procedures in an effort towards greater uniformity in serological test performance. The Sub-Committee on Serology and Laboratory Aspects of WHO (1949) considered its report (WHO/VD/38) an outline for the conference to be held in 1951 or 1952. This outline was approved by the fifth session of the WHO Executive Board (EB5/86 Rev.2) along with the other activities of WHO under the laboratory programme of the organization. The planning of the laboratory conference has since gone forward actively and Dr. Peter Krag, previously Director of the Serological Department of the State Serum Institute in Copenhagen, has accepted assignment with the World Health Organization to direct the conference, the site of which will be announced later.

Invitation.—Taking into account the continued use in all parts of the world of a great variety of sero-reactions, with often divergent results; the appearance of several new serodiagnostic tests which were not evaluated at previous international laboratory conferences; the development of purified

cardiolipin-lecithin antigens (Pangborn); the treponema immobilization reaction (Nelson) and the general need for determining the most useful and practical serodiagnostic methods for mass serological examination, the Director-General of the World Health Organization has the honour of inviting authors of methods for the serodiagnosis of syphilis to register preliminary applications for admittance to the WHO Serodiagnostic Laboratory Conference to be held after October 1951.

Procedure and Requirements. (*a*) Name, position, laboratory address of applicant, including personal and professional data, and lists of scientific publications to be sent to WHO accompanied by a statement indicating in detail the advantages offered by the special test and the laboratories presently employing the particular technique (as far as possible).

(*b*) Only an author-serologist, or a serologist designated by him, will be allowed to perform the author's procedure during the conference; each author-serologist can enter several reactions, but only one may be accepted.

(*c*) A suitable number of participants in the conference will be selected by WHO on the basis of the advice given by the Committee of WHO on Serology and Laboratory Aspects, scientific, technical and geographical, other relevant factors being taken into account.

(*d*) The preliminary applications to be received by WHO not later than 15 July 1950.

CORRESPONDENCE

International Sociological Association World Congress

To the Editor: It will interest students of Social Medicine that the first World Congress of the International Sociological Association is to meet this year to formulate a line of action for the betterment of international relations. I have just received an official notice from Mr. Erik Rinde, the Secretary of the Provisional Executive Committee (whose address is Grev Wedelspl. 4, Oslo, Norway), that the International Sociological Association with the United Nations Educational, Scientific and Cultural Organization, is now making preparations for a World Congress of Sociologists to be held in Zurich, Switzerand, from 4 to 9 September 1950.

This Congress of Sociologists has been arranged to take place simultaneously with the meeting of the International Sociological Association. Whereas the programme of the World Congress will be devoted primarily to scientific discussions of sociological problems of common interest, the meeting of the Council and the Executive Committee of the International Sociological Association will be concerned with the perfection of the organization established in Oslo, the election of an Executive Committee and officers to replace the provisional Executive Committee and officers elected at Oslo, the reports of committees and the consideration of a programme.

The general theme of the World Congress is *sociological research in its bearing on international relations*. The aim of the Congress is to re-establish professional contacts among sociologists of various countries of the world and to explore and advance the potential contributions of sociology to the objectives of world understanding and world integration.

Papers on topics and projects falling under this general head are being invited from countries all over the world. Papers may be of two kinds: either *original contributions* on one or more aspects of the problems at issue, or *analytical and critical surveys* of contributions made, research conducted or projects discussed in the respective countries or regions. The papers thus received may then be grouped around a series of sub-themes for round-table discussions at the Congress. Among such sub-themes have been suggested: nationalism, displaced persons' problems, immigrants and their adaptation, ethnic groups within nations and their influence on foreign policy, international communications, conditions of intercultural understanding, problems of international organization. It has also been found desirable that a special session be set apart for the analysis of the problems confronting Unesco in its efforts to develop social science research on an international level. Particular emphasis will be given to the discussion of the large-scale enquiry conducted into tensions affecting international understanding. The Congress will thus furnish a unique opportunity for thorough discussions among leading sociologists of the problems and potentialities of internationally co-ordinated research directly relevant to United Nations action.

Papers should be submitted before 15 July 1950 to Mr. Erik Rinde, the Provisional Secretary of the Association. Sociologists from all countries are invited to participate in the work of the Congress either as individuals, as members or as representatives of sociological organizations. They should notify the Secretary as early as possible, indicating the topics in which they will be particularly interested at the Congress.

The International Sociological Association was founded in Oslo to meet the long-felt need of sociologists in different countries for the exchange of information, co-ordination of education and research and of mutual contact. The Association is directed by a Provisional Executive Committee, with Professor Louis Wirth of the University of Chicago as President, and Professor Georges Davy of the University of Paris, and Professor Morris Ginsberg of the London School of Economics as Vice-Presidents.

All enquiries regarding the Congress should be directed to the Secretary of the International Sociological Association, Mr. Erik Rinde, Grev Wedelspl. 4, Oslo, Norway.

2, Barbican Building, Louis F. Freed.
President St.,
Johannesburg.
21 April 1950.

An Undescribed Fever

To the Editor: With reference to the cases referred to by Dr. Loewenthal in your *Journal* of 15 April, the symptoms and signs described by the writer fit in quite clearly with the condition amaas or kaffir-pox.

During my practice at Camperdown, Natal, I saw several cases of this disease, and they seem to correspond with the case described by Dr. Loewenthal.

A. A. Gordon.

Exchange Buildings, 89 Main Street
Port Elizabeth.
20 April 1950.

To the Editor: I should like to add another case to the list of those described by Dr. L. J. A. Loewenthal in the *Journal* of 15 April 1950.

The patient, a boy aged 2½ years, was referred to me by Dr. W. Harrison, of Roodepoort, as a very unusual skin condition which he had never seen before. The history given, when the child was first seen by me on 18 February 1950, was that a rash had suddenly appeared three days before and had not altered markedly since. The child had not seemed ill, but a rise in temperature to 101° F had been noted on 16 and 17 February. On the day of examination there was no fever and the child was lively and appeared in excellent general health. He had been vaccinated at 18 months and had not had chicken pox.

On examination there was a wide-spread pinkish-red papular and papulo-vesicular eruption. The lesions were discrete, up to 0.5 cm. diameter, and fairly closely set together on exposed parts, arms, legs and face. The trunk, back and front, had fewer and more scattered lesions. Vesiculation was not marked and close inspection was necessary to establish that vesicles were indeed present. There was obvious painless enlargement of the occipital, posterior cervical, axillary and inguinal lymph glands. No lesions of the buccal mucosa were noted. The liver and spleen were not enlarged.

The mode of onset and the appearance and distribution of lesions did not correspond with any previously described disease and I reported to Dr. Harrison that I was unable to make an accurate diagnosis but suspected that a virus might be the causative agent. I suggested that the child be kept under observation and given no treatment unless there were some new developments, in which case I advised giving aureomycin. There were no new developments, however, and Dr. Harrison found that the rash had disappeared at the end of a week.

James Marshall, M.D.

140 Lister Buildings, Jeppe Street
Johannesburg.
21 April 1950.